INTRODUCTION

The purpose of this article is to introduce the emerging specialization of "cognitive neuropsychiatry" to practicing neuropsychiatrists, and in particular those outside the United Kingdom. Cognitive neuropsychiatry is a new hybrid discipline that tries to apply some of the successful methodology of its sister discipline, cognitive neuropsychology, to (neuro-) psychiatric disorders.\(^1\)\(^2\)

The main starting point is cognitive neuropsychology, or to go back even further, neuropsychology. In fact, the latter term is fairly modern, having been used initially in 1913 but only really becoming established after a publication by Donald Hebb in 1949 and the launch of the journal *Neuropsychologia* in 1963.\(^3\) There is a reciprocity to these neurological
branches of medicine and psychology. In one direction, neuropsychology seeks to determine the relevance of brain damage or disease to changes in behavior and cognition. In the other direction, patients presenting with a set of behaviors or cognitive deficits, or even a specific deficit, may be studied and tested by the neuropsychologist in order to aid in diagnosis. It may be possible to say whether the behavior or deficit in question resembles those seen in circumstances where a lesion or disease is known to have had an effect.

The same reciprocity is inherent in cognitive neuropsychiatry, which takes full advantage of the established bedrock of knowledge about brain-behavior-cognition relationships. What marks the approach as distinctive within medical specialisms is the explicit move beyond diagnosis and classification, toward offering a cognitive explanation for the disorder and, as an important second, location of the brain systems responsible. The characteristic feature of cognitive neuropsychiatry, however, remains its theoretical approach: using patterns of impaired and intact cognitive performance found in patients to inform and revise current models of normal cognitive functioning. A few examples of this will be given below. The relative success or failure of these examples rests on their heuristic value, their testability, and the degree to which they are supported by converging evidence from clinical neuropsychology and cognitive neuroscience.

The remit of cognitive neuropsychiatry is broad—arguably broader than neuropsychiatry or behavioral neurology, although firm boundaries have yet to be drawn. For example, schizophrenia is not normally regarded as falling within the ambit of the neuropsychiatrist, despite the increasing evidence of its neurological basis. The reason for this is in part a matter of pragmatism; with a lifetime prevalence of approximately 1%, the disorder will always be part of mainstream psychiatry. Furthermore, there are no psychiatric disorders that cannot, in principle, be conceived to have a neurophysiological basis, and many that are associated (albeit subtly) with altered neuroanatomy. Nevertheless, schizophrenia is a core interest in cognitive neuropsychiatry, as we shall discuss below.

Another important disorder is Alzheimer's disease. In the United Kingdom, presenile cases tend to be managed by neurologists, occasionally with assistance from a neuropsychiatrist—especially where diagnosis is obscure and comorbid depression and alcohol misuse complicate the picture. After age 65, patients may be managed mostly by geriatricians, internists, and psychogeriatricians, as well as, of course, family practitioners. Again, the neuropsychiatrist in his or her hospital base does not usually have the community and social resources to manage such patients, who at this age may be classed as having a common disorder. However, here too the neuropsychiatrist may be called upon to assist with diagnostic and some management issues.

So what of cognitive neuropsychiatry? It is the phenomenological manifestations of schizophrenia, Alzheimer's, and the rest that comprise the "bread and butter" of cognitive neuropsychiatry—delusions, hallucinations, misidentifications, and apparently extraordinary
behaviors. By seeking analogies from less complicated and better understood neurological disorders, cognitive neuropsychiatry tries to formulate a functional model or context of the symptom. Note the insistence on *symptoms* (occasionally symptom clusters) rather than diagnoses per se. This is another departure from mainstream neuropsychiatry. The argument here is that "causal" cognitive explanations are not, and indeed cannot be, relevant to *all* the manifestations of disorders and diseases—such as cerebral atrophy or hemiparesis—although such manifestations may be important for the diagnostic process.

**DELUSIONS**

Given the reliance on analogy, it is hardly surprising that the psychiatric phenomena that to date have proved most amenable to the cognitive neuropsychiatric approach are those demonstrating the greatest "organicity"—for example, the delusional misidentification syndromes. Indeed, Ellis summarizes work in this area as "an exemplary vindication of the new discipline." This work was partly experimental and partly theoretical, and was able to place such phenomena as the Capgras delusion (where the patient believes that a relative has been replaced by impostors), and the Fregoli delusion (where the patient believes that a certain person has assumed different physical appearances) within the cognitive framework of normal face processing, of which the anatomical counterpart is fairly well understood. Out of this framework it was formulated that such patients, although able to recognize faces, have an abnormal sense of familiarity. This sense is normally conveyed via a ventral brain pathway in the form of an orienting or arousal response. Conventional tests of facial recognition were relatively intact in such patients, offering supporting evidence, but a more critical test was that autonomic responses to familiar and unfamiliar faces were no different. Hence if a patient is confronted by a person whom he or she perceptually recognizes and yet does not experience the usual reassuring feeling of familiarity, this can lead to an interpretation that assumes the person is an impostor. This last step of attribution requires further explanation and may have its roots in reasoning and judgment processes or in individual biases.

Although it is unlikely that a unified theory of all delusions will be forthcoming in the near future, it is desirable that theories of normal belief formation should eventually cast light on both the content of delusions and the processes whereby beliefs are formed.

**HALUCINATIONS**

Hallucinations are common to both
"functional" and "organic" disorders. Hence one place to begin the search for an underlying mechanism is in conditions with known pathophysiology. In the case of auditory hallucinations, such conditions are less apparent, although studies of epilepsy and brain stimulation form a reasonable point of departure. The notion that "irritation" of auditory association areas may produce such experiences has now been put to the test with the advent of functional neuroimaging techniques, and indeed finds support. Further work is still required to explain the particular grammatical construction and emotional content constituting most auditory hallucinations.

Visual hallucinations have been seen as the province of neuropsychiatry ever since the discipline emerged. Why visual experiences should be induced more often than auditory experiences by brain perturbation remains an intriguing question. We are now well on the way to a comprehensive explanation of the phenomena that takes into account the role of the sleep-wake cycle, neurotransmitter imbalance, degradation of sensory input, and the constructive capacity of the human visual system. Further work contrasting neuropsychological deficits in people with neuropsychiatric conditions with and without hallucinations is awaited with interest. The extent to which discrete psychopathologies such as hallucinations can be mapped onto physiological processes remains an active area of investigation.

**INSIGHT**

Insight and awareness is a crucial area with theoretical as well as practical implications. Several authors have proposed an analogy with anosognosia, the lack of awareness of physical deficits sometimes observed in patients following right hemisphere lesions. Lack of awareness of behavioral and personality change following brain injury and degenerative disorders can put a huge burden on caregivers and pose major challenges to clinical management. Careful content analysis of statements made by patients recovering from anosognosia can be used to test hypotheses about the origin of the deficit. In tandem with this effort, fractionation of the hitherto vague general psychiatric concept of "poor insight" into components that may be more likely to have a neurocognitive basis (such as the ability to relabel certain symptoms as pathological) and those whose basis may lie in cultural and social models of disease (such as whether or not one is suffering from an illness) is likely to render the problem tractable.
THOUGHT AND LANGUAGE DISORDER

The disjointed, and at times incomprehensible, speech of patients with schizophrenic thought disorder has long intrigued neuropsychiatrists who saw similarities with certain kinds of fluent aphasia. Similarly, the poverty of speech commonly observed in patients with negative symptoms brings to mind the lack of spontaneity and alogia of the frontal lobe patient. Linguistic analysis of schizophrenic speech has attracted polarized opinions. However, the distinctions between phonology, lexical-semantics, and pragmatics of language have been applied to schizophrenic thought disorder and suggest problems in making use of context online to understand, and by inference plan, utterances. Others have concentrated more on semantics and have taken the techniques of semantic priming from the experimental psychology laboratory into the clinic. The work is based on the view that our storage of word meanings is in the form of a network of nodes, whose proximity is determined by the degree to which they share meaning. The exposure to a given word leads to the automatic spread of activation through the network, from one node to its neighbors. Spitzer has argued that in schizophrenic thought disorder this spread is broader and leads to activation of words of only indirect semantic relatedness. This "spills out" into conversation as thought disorder.

AFFECT AND COGNITION

Psychologist Martin Harrow once quipped that patients don't get admitted to the hospital because of bad grammar! It is the content—as well as the form—of the thought disorder that leads to concern. Indeed, the discussion of emotive personal themes has recently been shown to exacerbate thought disorder.

A crude distinction between the subject matter of neuropsychology and neuropsychiatry used to be made on the basis of affect. That is to say, the deficits (and excesses) of neurological patients were seen as value-free and content-neutral. The disorders of psychiatry, on the other hand, were viewed as essentially about topics involving personal investment, beliefs, and desires. The advent of "affective neuroscience" has swept such distinctions away. Thanks to the work of Le Doux and Damasio, emotions are once again a legitimate topic for research. Again neuroimaging has boosted this reappraisal with specific neural substrates for different (basic) emotions being mapped out: the amygdala for fear, the insula for disgust, and possibly the orbitofrontal cortex for anger.
The neurobiology of affective disorder has, of course, always been a major focus of research, as have the behavioral manifestations of lesions of the limbic system. This is outside the scope of the current article. Interest has now broadened to encompass disorders such as autism, now regarded as being a disorder of social cognition in which the natural ability to infer another's mental state is impaired. Depersonalization disorder is another neglected condition, which has long had its "organic" and "functional" proponents. Advances in the understanding of the neurobiology of emotional controls, and the way in which affect colors cognition, have led to suggestions that such patients fail to experience (as opposed to recognizing) emotional arousal. A model comprising reduced subcortical activation in the face of emotional stimuli and reduced physiological arousal, combined with intact frontal subsystems that are in inhibitory mode, can be pieced together from the growing body of evidence. The interaction of similar networks has been proposed to account for certain cases of psychopathy. The additional element appears to be damaged frontal subsystems, which lack inhibitory tone. While it is foolish and undesirable to seek to "explain away" complex biopsychosocial phenomena, advances in the cognitive neuropsychiatric understanding of the whole range of disorders add an important perspective.

**CONVERSION DISORDER**

Conversion hysteria is a famous battleground for the proponents of exclusively psychological versus physiological modes of mental states. The crux of the problem lies in the philosophical conundrum of having to explain how psychological or mental states (caused, in this case, purportedly by a reaction to an excessive emotional state) can produce specific long-term motor, sensory, and cognitive effects in subjects who claim not to be consciously responsible. In terms of the history of ideas, "hysteria" is an object lesson. Although Freud's work drew attention to the clinical disorder over a century ago, there has been little or no empirical research concerning the neural and psychological mechanisms. Freudian formulations have not endured, and the view that all hysterical conversion is undiagnosed neurology has suffered a similar fate. Into this void, cognitive neuropsychiatry has begun to take its first tentative steps. This is not so brave (or foolish) as it might appear. Conceptual advances in implicit and explicit cognitive influences, the understanding of mechanisms of dissociated awareness, and evidence provided by functional neuroimaging on the brain areas for willed intentions have all made such a venture feasible.

Although only a handful of reports have previously attempted to explain hysterical behaviors in terms of neuropsychological disorders, the nature of the condition and the brain structures responsible have not been significantly elucidated beyond views expressed at the end of the
nineteenth century in the work of Janet (1889), and Freud (1894). Given that hysterical paralysis, like normal voluntary movement, must express itself through the structural medium of normal brain functioning, the emergence and availability of noninvasive neuroscience methodologies such as functional imaging has provided researchers with the possibility of finding out which areas of the brain are involved. A recent case study employing PET provides some insights as to the abnormal physiological brain activations in hysterical motor conversion.49

Another phenomenon that has been classed both as a conversion disorder and a neurological deficit is pure retrograde amnesia. The careful analysis of certain cases of "fugue" has shown that it is just as possible to uncover positive evidence of intractable social and personal dilemmas as it is to uncover a neurological lesion, provided the effort is made. The weakness of cognitive neuropsychiatric explanations for this syndrome has been the lack of a convincing cognitive account of retrograde amnesia, given current theories of memory. This weakness is beginning to be addressed50 as such theories (rooted in functional neuroanatomy51) begin to accommodate such case material.52

FUTURE PROSPECTS

We have discussed above just a few examples of the territory in which cognitive neuropsychiatry is beginning to apply itself successfully. Many other conditions and topics have benefited from this approach (e.g., posttraumatic stress disorder53 and obsessive-compulsive disorder54), and no doubt more will benefit in the future. Evidence of the growing interest in this field can be seen from the growing numbers of recent books,35,55,56 together with the launch in 1996 of the new quarterly journal Cognitive Neuropsychiatry. We would like to see more activity in developmental disorders and more cognitively motivated applications to rehabilitation. Also, there is a need to take stock of functional and structural neuroimaging so that these powerful technologies can be put to the service of theoretical understanding and not merely cerebral localization.57 As we leave the "Decade of the Brain," we predict that it will be the combined use of multimodal technologies and careful clinical observation that will provide the greatest advances in theoretical and practical neuropsychiatry.

REFERENCES

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